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BURN SHOCK IN DOGS AND THE EFFECT OF NEUROPLEGIC
SUBSTANCES ON ITS COURSE

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BURN SHOCK IN DOGS AND THE EFFECT OF NEUROPLEGIC
SUBSTANCES ON ITS COURSE

Following is the translation of an article by
Ye. M. Gindin, O. L. Pokrovskaya, and L. V. Lebe-
deva entitled "Ozhogovyy Shok u Sobak i Vliyaniye
Na Ego Tsecheniye Neyroplegicheskikh Sredstv"
(English version above) in Khirurgiya (Surgery),
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From the chair of surgery (Chief -- Prof. A. S. Rovnov)
at the Central Institute for the Advanced Training of Physi-
cians and the Laboratory of Experimental Physiology on the
resuscitation of the organism (Head -- Prof. V. A. Negovskiy)
of the Academy of Medical Sciences USSR.

In spite of a considerable quantity of clinical and
experimental investigations, many questions of the patho-
genesis, clinical aspects, and therapy of burn shock have
not received sufficiently complete solution up to the present
time.

The object of the present work is the study of the
clinical peculiarities of the course of burn shock in dogs
to obtain a more adequately based solution of the questions
of its therapy.

The maximally widespread methods of experimentally
inflicting burn by boiling water, alcohol flame or blowtorch
possess a number of substantial disadvantages, which do not
in all cases permit the burn trauma to retain a constant area
of the burn surface, exposure, and temperature of the thermal
agent. The free method of inflicting burn, proposed by V. N.
Zhizhin and used by us in the present work, is free from
these disadvantages from our point of view.

The experiments were set up on healthy dogs 12 to 14
kg in weight. The area of the burn surface amounted to 15
percent of the body surface. To calculate the area of the
body surface we used the formula of May: $S = K$ times the cube
root of T^2 , where S is the area of the body surface, K is a
coefficient, T is the weight of the animal. The coefficient
 K , expressing the ratio between the area of the body surface
and the weight of the animal, was determined by V. N. Zhizhin

experimentally and is equal, approximately, to 9.3 for a dog 12.3 kg in weight; to 9.9 for 14.3 kg; to 10.3 for 15.2 kg, etc. On the shaved skin of the anterior surface of the thorax and the abdomen, a trapezium was drawn, the area of which amounted to 15 percent of the area of the body surface. The burn was applied to this surface. The other parts of the body were protected by a sheet. The regions of the heart and the sexual organs as the maximally shockogenic, were additionally covered with a layer of wadding. We assembled a special installation for inflicting the burn by steam of a definite temperature, consisting of an autoclave, a rubber hose, a copper pipe coil, and a blowtorch. The steam pressure in the autoclave reached two atmospheres. The discharge stopcock of the autoclave was joined by a rubber hose with the copper coil, made of copper tubing one m in length and 8 mm in diameter. The coil was fastened to the burner of a blowtorch, by the flame of which the steam was headed, passing along the coil from the autoclave. The temperature of the steam at the site of contact with the skin was equal to 110-120°. The burn was inflicted for the course of 15 minutes. The exposure was diminished in the case of a sharp fall in the animal's arterial pressure.

At the time of the experiment the arterial pressure and the respiration were registered on the kymogram, observations were made of change of the venous pressure, the rectal temperature, pain, eye, and tendon reflexes. In blood samples taken from the marginal vein of the ear before the burn and 15, 45, 90 and 180 minutes after the burn, a count of the erythrocytes, a determination of the hemoglobin and the volume index of erythrocytes (with the aid of the hematocrit), were conducted.

Burn trauma was inflicted on 12 dogs. In the period of preparation for experiment, six of them were found in a state of excitation. The initial arterial pressure in the animals was in limits of from 138 to 205 mm of mercury, the venous pressure -- from 60 to 100 mm of water. The pulse frequency was from 70 to 200 beats per minutes, the respiration -- from 13 to 90 per minutes. In the excited animals the indicated indexes as a rule were higher.

We observed a definite phasicity in the disturbances of the physiological functions during the inflicting of the burn trauma.

In all the animals the initial reaction to burn was expressed in an extraordinarily strong motor excitation accompanied by micturition or defecation, by a rise in the arterial pressure to 190-340 mm and of the venous pressure above 300 mm; the pulse frequency and the respiration were diminished in the first minutes by almost two times in comparison with

the initial values. In the presence of continued application of the burn the dogs were still found in a state of excitation; its intensity was, however, gradually lowered. More pronounced paroxysms of excitation were renewed when the jet of steam came in contact with as yet unburned areas of skin. At this time the arterial pressure was briefly raised. The level of arterial and venous pressure, considerably exceeding the initial figures, had at the same time a certain tendency toward lowering. On the second to the sixth minute there ensued a sharp increase in the frequency of the pulse to 100-160 beats per minute. The respiration was also increased in frequency. The rectal temperature was raised by 2-5°, reaching 41-44.

According to the literature data (I. R. Petrov, S. I. Banaytis, P. A. Nalivkin, S. G. Frid, F. I. Kovshikov, G. L. Frenkel' and G. F. Koveshnikova and others), the reactions described are characteristic for the erectile phase of burn shock. However the changes in the intensity of the reactions of animals to burn trauma which we observed in this period are evidence, apparently, of a series of stages in the course of the erectile phase of burn shock.

In our experiments the duration of the erectile phase was short (7-14 minutes), which coincides with the observations of other authors (B. N. Postnikov, G. L. Frenkel', and others).

The transition to the torpid phase had already begun in the period of inflicting the burn trauma. The first signs of the development of the torpid phase were changes on the part of the central nervous system. From the seventh to the 14th minutes from the beginning of the burn, the paroxysms of motor excitation ceased. From this moment the animals lay completely immobile, reaction to sound and pain stimuli was absent. In certain animals the pupil reflex disappeared; the eye reflexes were retained.

Subsequent signs which characterize the transition to the torpid phase were secondary increase in the frequency of the pulse (to 200 beats per minute and more) and an intensification of dyspnea (to 200 inspirations per minute and more). The arterial pressure at the moment of terminating the burn was close to the initial value - 122-205 mm. The level of the arterial pressure was more significantly lowered in individual animals - to 24% in relation to the initial value.

Thus, in the period of inflicting the burn trauma, shock developed in all the animals. The severity and duration of the shock state depended on the individual sensitivity of the animals.

In some animals the shock state lasted for minutes, in others for hours, in a third group -- for tens of hours.

This provided a basis for dividing the experiments into three groups.

The animals of the first group (six dogs) perished shortly after the cessation of the burn in periods of from one minute 50 seconds to 19 minutes 50 seconds. After this period the arterial pressure fell critically, dyspnea and tachycardia increased (Fig. 1.). The cessation of the cardiac activity and respiration ensued after a brief agony, accompanied by decerebrate rigidity. A peculiarity which distinguished the animals which perished several minutes after trauma from the others was their extremely excited state before experiment and the maximal lowering of the arterial pressure at the end of infliction of the burn.

The animals of the second group (two dogs) perished three hours 45 minutes to four hours 45 minutes after infliction of the burn. In the observations for change of the state of the animals in the course of the torpid phase of burn shock one could note three stages of its development (Fig. 2).

The first stage had already begun at the time of the burn and continued 10-25 minutes after its termination. The dogs lay immobile. They did not react to sound and pain stimuli. The pupil reflexes were absent. Dyspnea and tachycardia increased. The arterial pressure was lowered, but did not, however, fall below 100-120 mm. The slow lowering of the arterial pressure was evidence of a more favorable course of the burn shock and of the possibility of transition to the following stage, characterized by a temporary improvement in the state.

This stage combines in itself signs both of the erectile, and also of the torpid phases. E. A. Asratyan observed a similar stage in the presence of the development of traumatic shock and designated it as the stage of depression. This name, correctly determining the pathogenetic essence of the processes which are breaking out, should also be retained for burn shock.

The first sign of the transition to the second stage of the torpid phase of burn shock was the stabilization or rise in the arterial pressure to 130 mm. Then the dyspnea was gradually diminished and the pulse was retarded, reaching the initial magnitudes after one to one and a half hours. The rectal temperature was also slowly lowered. In the last place changes ensued on the part of the central nervous system -- the reaction to sound and pain stimuli was restored, the animals raised the head, looked around at the sides, paroxysms of motor excitation were periodically noted.

This second stage was characterized by a temporary compensation of functions. The presence of such a stage both in the presence of burn shock, and also of traumatic shock

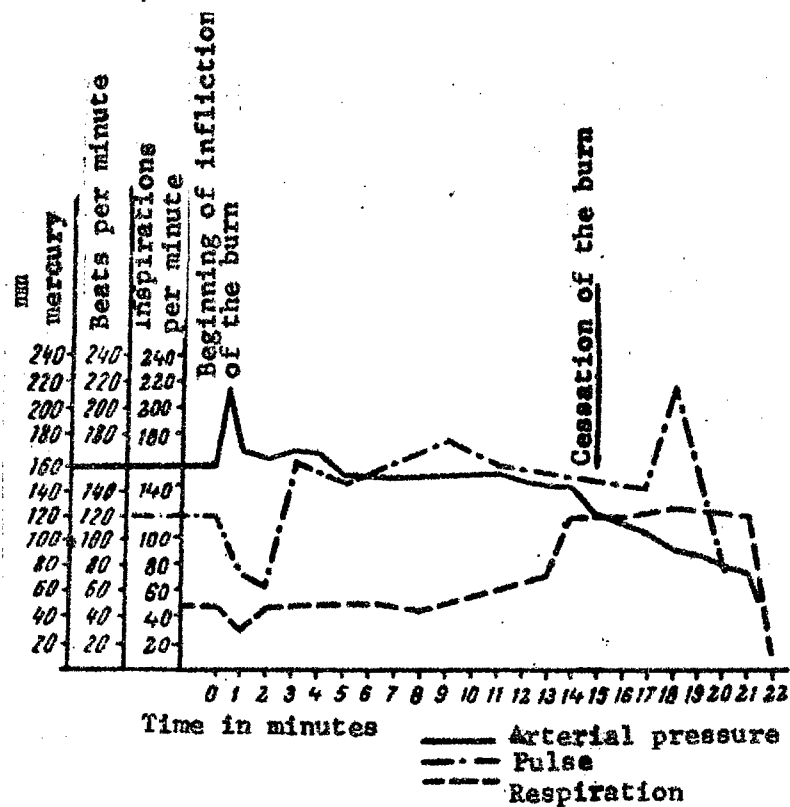


Fig. 1. Changes in the arterial pressure, pulse, and respiration during the infliction of burn trauma and in the post-burn period in animals of the first group.

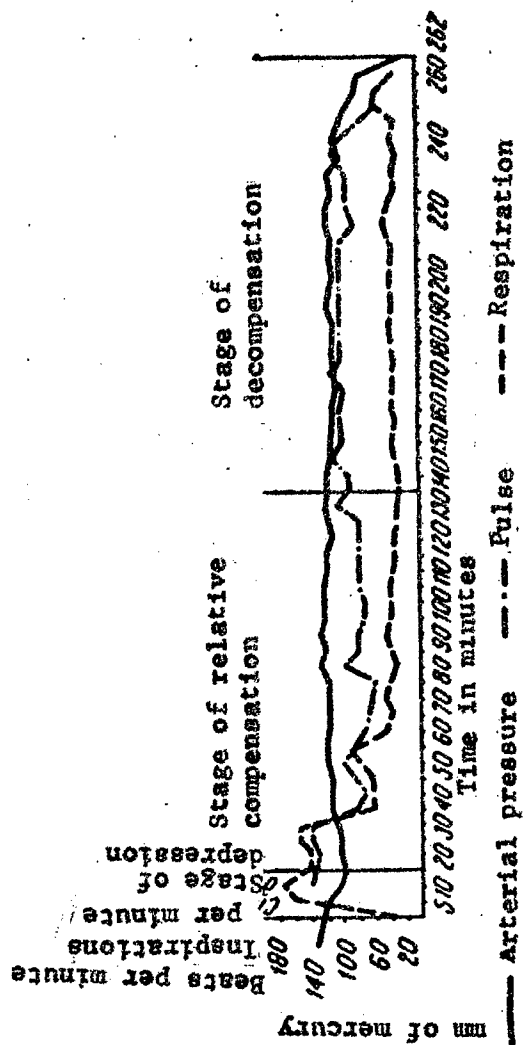


Fig. 2. Changes in the arterial pressure, pulse, and respiration in the post-burn period in animals of the second group.

has been noted by many investigators (B. N. Postnikov, G. L. Frenkel', P. A. Nalivkin, and Vasil'kovan, E. A. Asratyan, and others). In our experiments this stage lasted around two hours, after which the state of the animals worsened again.

The first sign of the transition to the third stage of the torpid phase was the disappearance of reaction to external stimuli, a general depressed state of the animal. The pulse frequency was increased anew to 120-240 beats per minute. The respiration remained infrequent. The arterial pressure, being lowered gradually, did not fall, however, below 90-100 mm. An acute lowering of the arterial pressure ensued 25-30 minutes before the cessation of the cardiac activity. In this period agony developed. The rectal temperature remained above normal even in the agony period. In contrast to the data of many authors (N. N. Burdenko, B. N. Postnikov, G. L. Frenkel', V. Avadkov, and others), we did not observe the early development of hypothermia in the presence of burn shock. The duration of the third stage did not exceed one and a half to two hours in animals of the second group.

The third stage of the torpid phase should be considered as the stage of decompensation of functions (B. N. Postnikov, G. L. Frenkel', P. A. Nalivkin and Vasil'kovan, E. A. Asratyan, and others).

The animals of the third group (four dogs) perished 16-36 hours after the infliction of the burn trauma. Three stages were noted in the development of the torpid phase of burn shock, just as also in animals of the second group: depression, relative compensation of functions, and decompensation (Fig. 3).

In contrast to the second group, in dogs of the third group the stage of relative compensation of functions is distinguished by a considerably greater duration -- up to 15-35 hours, and by a more complete emergence from the shock state. The animals walked, reacted to caresses, ate much and greedily. An acute worsening in the state ensued 20-30 minutes before death.

In accordance with the data of many authors (Yu. Yu. Dzhanelidze, B. N. Postnikov, I. D. Zhitnyuk, and others), but in contrast to the data of I. R. Petrov, an early and long lasting growth in the hemoconcentration and hemolysis was observed in the presence of burn shock. Fifteen minutes after the burn, the quantity of hemoglobin was increased by 5-34 active units, the erythrocytes -- by 210,000 - 1,400,000, the volume index increased by 2-14% in comparison with the initial data. A further increase in the hemocncntration was traced in the course of three hours after the burn.

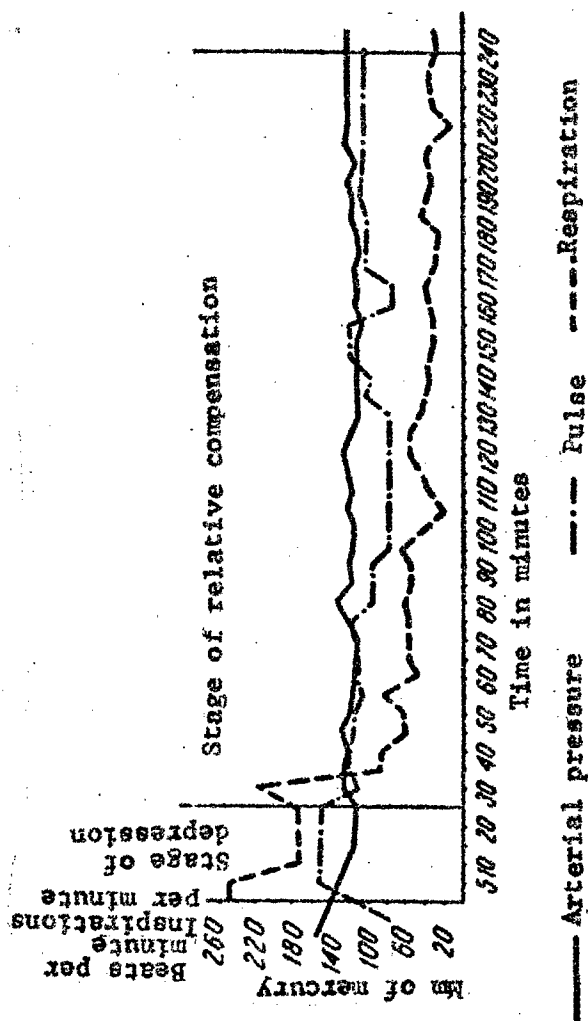


Fig. 3. Changes in the arterial pressure, pulse, and respiration in the post-burn period in animals of the third group.

In the animals which perished from burn shock in the region of the burn a dense dark-brown coloration of the skin, edema of the subcutaneous fatty cellular tissue and of the muscles were determined macroscopically, which corresponded to a burn of the third degree.

An acute, congestive plethora of all the internal organs and the presence of a pronounced hemolysis in the vessels appeared to be dominant in the morphological picture of the animals which perished after burn trauma.

In conclusion it should be noted, that the dynamics of the change of functions has particularly important significance in the clinical picture of burn shock.

The first signs which give evidence of a transition to a more severe stage, were changes on the part of the nervous system (general sluggishness, the disappearance of reactions to sound and pain stimuli). Tachycardia and dyspnea arose later. Dyspnea was absent in the stage of decompensation.

As our observations have shown, in agreement with the literature data (A. A. Troyanov, E. A. Asratyan, S. G. Frid, F. I. Kovshikov, B. N. Postnikov, and G. L. Frenkel', I. R. Petrov, and others), the retention for a long time of an arterial pressure equal to the initial pressure or even exceeding it, is characteristic of burn shock. On the basis of this fact B. N. Postnikov and G. L. Frenkel' assert, that the level of the arterial pressure cannot be a leading index of burn shock. Our experimental data do not permit us to completely agree with this. If the absolute magnitudes of the arterial pressure actually have a relative value, then the dynamics of its change appears to be a leading sign for the determination of the severity of the state. The slow lowering of the arterial pressure is evidence of a transition to a more severe stage of shock, and death ensues in the presence of a rapid lowering. The stabilization of the pressure is characteristic of the stage of relative compensation of functions.

Thus, observation of the dynamics of the change of the functions permits us to distinguish two phases of shock: an erectile phase and a torpid phase. The torpid phase in its turn consists of three stages: depression, relative compensation of functions, and decompensation. The determination of the stage of development of the shock can serve as an indication for the use of one or another method of treatment.

In the second part of the work we have placed before ourselves the task of elucidating the action of neuroplegic agents on the course of the various stages of the torpid phase of burn shock, without using any other antishock measures under

these conditions.

To create an artificial hibernation we used a mixture, consisting of: 1) aminazine in a dose of 5 mg per one kg of body weight; 2) dimedrol (in the last experiments replaced by ethisine in the same dosage) - 5 mg/kg; 3) promedol or pantopon - 2 mg/kg; 4) atropine sulfate in a quantity of 0.3-0.6 mg per one animal; 5) vitamin B₁ - 50 mg per one animal; 6) pyramidone - 10 mg/kg [correcting misprint mk/kg] of body weight; 7) glucose - 250 ml of a 5% solution. The mixture was administered to the animal intravenously by a drop method.

To elucidate the effectiveness of the action of the neuroplegic mixture, three series of experiments were conducted on 22 dogs.

In the first series we studied on seven dogs the action of artificial hibernation on the organism of healthy animals (Fig. 4).

From the moment of administration of the first doses of the neuroplegic mixture the animals fell into a drowsy state, passing into sleep. The pain reflex disappeared in five dogs on the 6-23rd minute after the beginning of the administration of the mixture, it was weakened in two others. The temperature in the rectum was lowered by 3°. The arterial pressure was gradually lowered; at the end of the administration of the mixture it was equal to 77-118 mm, i.e., was 20-50% below the initial pressure. The venous pressure was not changed in six dogs, but was lowered from 200 to 60 mm in one dog. The pulse frequency at the time of administration of the mixture was not changed in some dogs, tachycardia developed in others. The frequency of respiration was also not changed in four dogs, a decrease in the frequency of respiration ensued in three.

After the termination of the administration of the mixture, which lasted from 22 to 103 minutes, the dogs were found in a drowsy state; the arterial pressure remained at the same level as at the time of administration of the mixture, the frequency of respiration, of the pulse, the rectal temperature were not changed. After two to four hours all the dogs gradually emerged from the state of hibernation.

On the following day the state of the animals was satisfactory, only a reduction in appetite was noted. In the following days the experimental animals were not different from healthy animals.

Thus, the intravenous administration of a neuroplegic mixture evoked in all the dogs a state of artificial hibernation, which did not leave after it any visible complications.

In a second series of experiments the course of burn

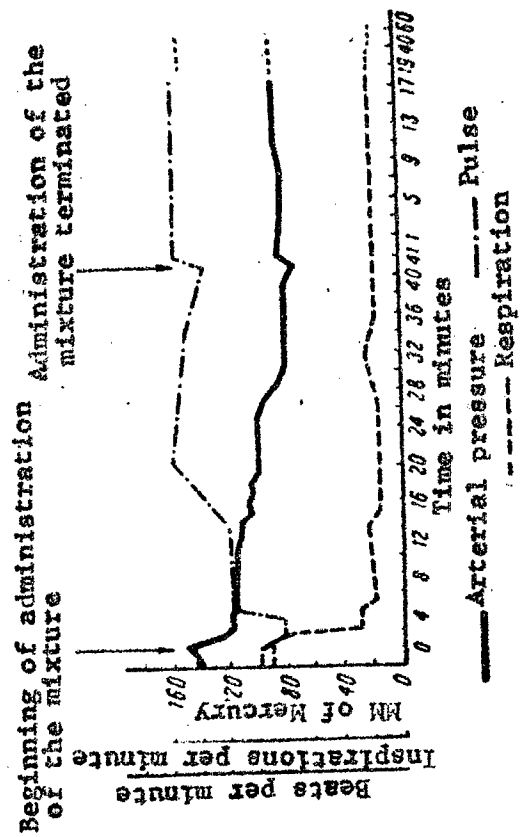


Fig. 4. Changes in the arterial pressure, pulse, and respiration in the presence of the administration of a neuroplegic mixture to healthy dogs.

shock in preliminarily hibernated animals was studied in four dogs.

After a state of hibernation had been created in the animals, burn trauma was inflicted on them for the course of 13-15 minutes. Under these conditions the dogs rapidly emerged from the drowsy state, and became very uneasy: whined noisily, barked, periodically manifested paroxysms of motor excitation. It should, however, be noted, that the motor excitation in these animals was considerably less pronounced, than in the control animals. The pain and pupil reflexes did not disappear at the time of the burn.

From the moment of inflicting the burn, the arterial pressure began to rise and the very highest rise -- 140-200 mm -- was reached on the fourth to the fifth minute. Then the arterial pressure was gradually lowered. However, when the infliction of the burn was terminated, it remained 5-28 mm above the initial level in three dogs. Dyspnea appeared on the sixth to ninth minute. The temperature in the rectum rose at the time of the burn by 1.5-5.3° above the initial temperature, but at the same time did not exceed 40.3°.

After termination of the burn, two dogs perished after 3 minutes 25 seconds and 31 minutes 25 seconds, a third -- after 8 hours, and the fourth -- after 36 hours. The course of the post-burn period was characterized by those same peculiarities in the preliminarily hibernated animals as in the control animals.

Thus, both at the time of inflicting the burn, and also in the post-burn period, the course of burn shock in the preliminarily hibernated animals was very little different from its course in the control animals. This difference was expressed in a lesser intensity of motor excitation, in a slower rise in the arterial pressure, and in its more gradual lowering.

The action of artificial hibernation on the course of burn shock was studied by us in a third series of experiments on 11 dogs.

In four dogs the administration of the neuroplegic mixture was begun in the stage of depression of the torpid phase of burn shock, i.e., 5-10 minutes after termination of the burn (Fig. 5). The state of the dogs in this period was severe: they lay immobile, pain and pupil reflexes were absent, the tendon reflexes were curtailed. The arterial pressure was equal to 100-160 mm, i.e., was 10-23% lower, than at the moment of termination of the burn; the pulse remained frequent - 186-260 beats per minute, the frequency of respiration reached 180-260 expirations per minute.

Several minutes after the beginning of the administration of the mixture of neuroplegic substances, the dogs fell

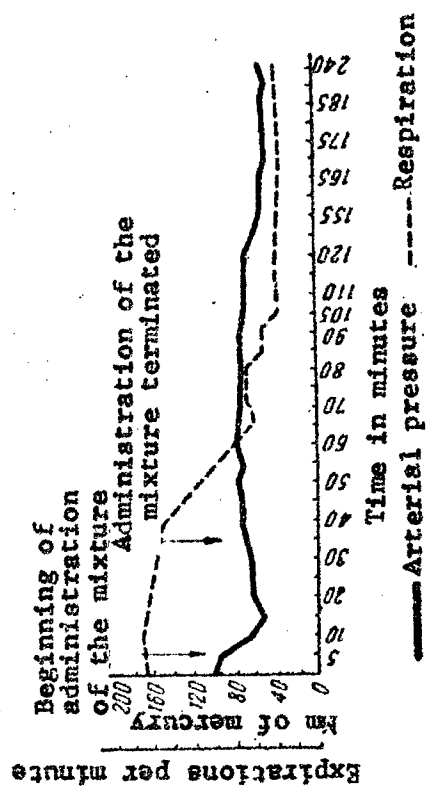


Fig. 5. Changes in the arterial pressure and respiration in the presence of the administration of a neuroplegic mixture at the stage of depression of the torpid phase of burn shock.

into a drowsy state. The arterial pressure began to be lowered rapidly. In the first minutes (5-15) it reached 60-80mm, i.e., was lowered 35-50 percent in comparison with what was observed before the administration of the mixture. In subsequent minutes the arterial pressure remained stable up to the termination of administration of the mixture. The frequency of respiration and pulse were hardly changed at all. The venous pressure was raised from 60-70 mm to 105-120 mm. During the time of administration of the mixture, the rectal temperature was gradually lowered to 39-41°, i.e., by 0.5-3°.

After termination of the administration of the neuroplegic mixture, which lasted from 20 to 50 minutes, the dogs remained in a drowsy state: they lay immobile with semiclosed or closed eyes, the pupils did not react to light, the pain reflex was absent, the arterial pressure was stabilized at low figures, the respiration was reduced in frequency to 40 expirations per minute. The pulse frequency was gradually diminished, the temperature at the end of the experiment was lowered 2-4° more.

Three dogs, which did not emerge from the state of hibernation, perished 13-26 hours after the inflicting of the burn; one dog emerged from the state of hibernation after nine hours, got up on its legs, ate greedily. After 16 hours the dog moved, was unsteady on its legs, but rapidly tired and fell; drank much. In subsequent hours the state of the dog worsened, and it perished 36 hours after the experiment.

Thus, in spite of artificial hibernation, the animals perished in the same periods as the control animals.

In the case of three dogs the neuroplegic mixture was administered in the stage of compensation of functions, 20-55 minutes after the inflicting of the burn (Fig. 6). At this time an appreciable improvement in the state of the animals ensued: they reacted to external stimuli, raised the head. The pain and pupil reflexes were restored, the arterial pressure was stabilized or even raised, at the moment of administering the mixture it was equal to 125-140 mm. The frequency of respiration in some animals began to be diminished, in others dyspnea was observed. In all animals tachycardia was still retained. The drowsy state ensued in the animals in proportion to the administration of the neuroplegic mixture: the eyes were closed, they lay immobile, they did not react to a whistle.

Immediately after the administration of the neuroplegic mixture the arterial pressure began to be lowered. During the first 20-50 minutes it was lowered to 70-100 mm and remained at this level until the termination of the administration of the mixture. The venous pressure was not changed during this period. The respiration gradually became less

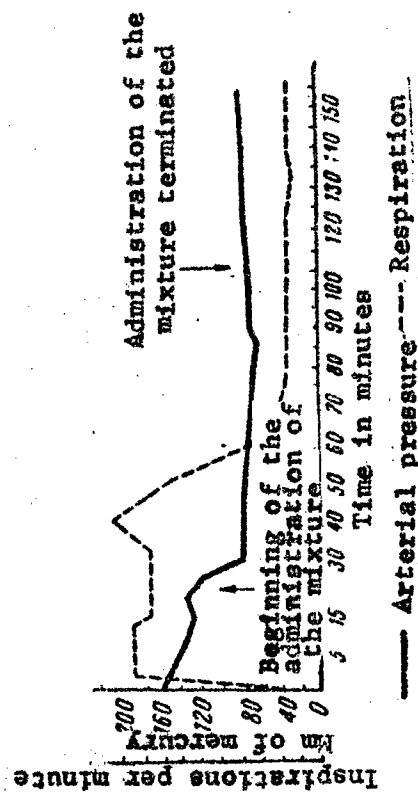


Fig. 6. Changes in the arterial pressure and respiration in the presence of the administration of a neuroplegic mixture in the stage of relative compensation of the torpid phase of burn shock.

frequent. The pulse remained frequent in the course of the entire period of administration of the mixture. The temperature in the rectum was lowered to $37-38^{\circ}$, i.e., by $1-4^{\circ}$.

The mixture of neuroplegic agents was administered in the course of 50-90 minutes. After termination of the mixture, the arterial pressure, the respiration, and the pulse were not changed in the course of the next 40-50 minutes. The temperature was still lowered by $0.7-0.9^{\circ}$ during this period.

Two dogs which did not emerge from the severe state, perished 22 hours after the inflicting of the burn. A third dog was active when observed after 16 hours, drank much and avidly. It perished after 24 hours.

Thus, the administration of a neuroplegic mixture in the period of a certain improvement in the state of animals -- in the stage of relative compensation of functions -- led them into a state of artificial hibernation, however, the animals perished in the same periods as did control animals.

To study the action of hibernation on the course of the third stage of burn-shock -- decompensation of functions -- we used four dogs in which burn shock had proceeded quickly and had ended in the control animals in their perishing in the first minutes after the burn (Fig. 7).

The state of these animals after the inflicting of the burn and before the administration of the mixture was extremely severe: the dogs lay immobile, in a state of complete prostration, with a drooping cyanotic tongue, and did not react to external stimuli. The pain reflex was absent in all dogs. The arterial pressure was quickly lowered.

From the moment of administration of the hibernation mixture, the state of the animals continued to worsen. The dogs did not react to external stimulation. The arterial pressure continued to be lowered rapidly, one to ten minutes after the beginning of the administration of the mixtures, agony ensued. The cardiac activity ceased 5-18 minutes after the inflicting of the burn trauma in the presence of the continuing administration of the mixture.

Thus, the use of artificial hibernation even in the stage of decompensation of functions did not give a positive result.

In the presence of investigations of the blood in dogs in the first hours after the administration of a hibernation mixture, no differences in comparison with the control animals were noted.

The pathomorphological changes in the hibernated dogs, which perished 13-22 hours after the inflicting of burn trauma, were the same, as in the control animals.

In comparing the clinical course of burn shock in control animals with the course of burn shock on a background of

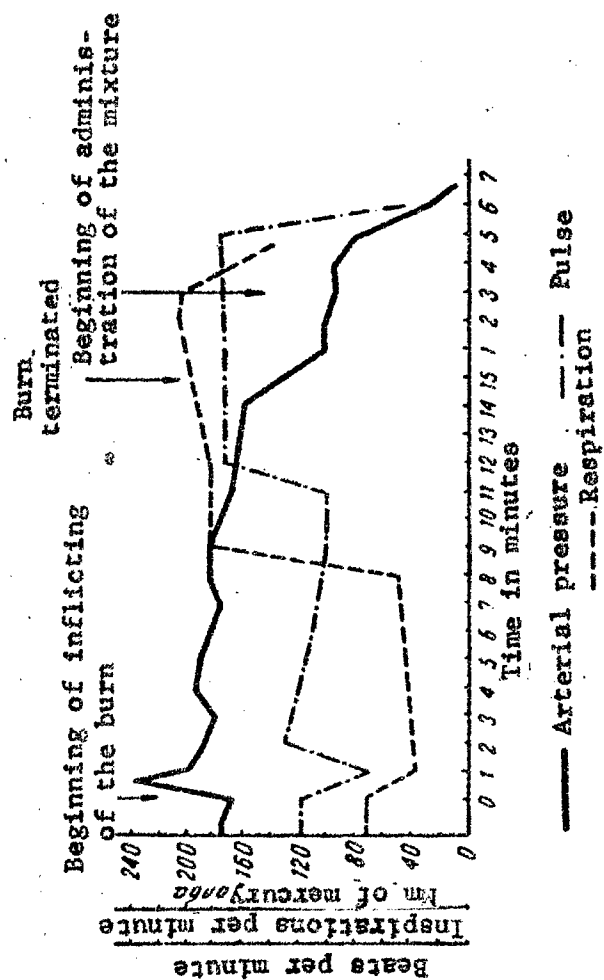


Fig. 7. Changes in the arterial pressure, pulse, and respiration in the presence of the administration of a neuroplegic mixture in the stage of decompensation of the torpid phase of burn shock.

artificial hibernation, one can note, that it facilitated only the removal of motor excitation, a lowering of the pain reaction, a removal of hyperthermy, a lowering, and then a stabilization of the arterial pressure, a reduction in the frequency of respiration and in a number of cases of the frequency of the pulse. However, such a favorable action of hibernation on the course of burn shock was temporary and did not influence the final result -- all the hibernated animals perished in almost those same periods after the infliction of burn trauma, as did the control animals.

Conclusions

1. Two phases of shock should be distinguished: the erectile phase and the torpid phase. The torpid phase consists of three stages: depression, relative compensation of functions, and decompensation.
2. The use of a mixture of aminazine, ethisine, atropine, promedol, pyramidone, vitamin B₁, and glucose, administered intravenously before the inflicting on the animals of burn trauma, did not exert a substantial prophylactic influence on the course and outcome of burn shock.
3. In dogs in the torpid phase of burn shock, hibernation -- applied in the stage of depression and relative compensation -- temporarily improved the state of the experimental animals. The use of hibernation in the stage of decompensation did not exert an influence on the course of burn shock.

END